REMARKS ON THE PATHOLOGY OF CHOREA.[1]

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IN an article in the "Lancet," 1889, Dr. A. E. Yarrod describes in cases of acute chorea of rheumatic origin, an increase of connective tissue in the cerebral cortex. This pathological condition accords with the clinical aspect of the disease, acute or chronic, as one of the characteristic features of chorea is the increase of movement on any excitation of the will. In other words, call the cerebrum into action by concentration on any special motor act, and as a result motor disturbance ensues.

Another marked symptom is the constant motor disturbance, which ends only with sleep, a fact significantly pointing to the cortex cerebri as the primary seat of the lesion in chorea. We have here to do with a disease whose principal symptom is an affection of the motor apparatus. What is clearly seen is that we have a loss of the normal inhibitory action of the cerebrum.

The constant motor disturbance in some acute cases is due to the irritant effect of the connective tissue—that is, if we accept Yarrod's theory; and this is probably very often due to a rheumatic diathesis.

In chronic chorea we have a lesion resembling that of multiple sclerosis, although less coarse in character. Diller, in the "American Journal of the Medical Sciences," December, 1889, well defines it as a fine general sclerosis. We may have patches of degenerated nerve-tissue, the result of diseases of the vessels, causing anæmia and interference with the nutrition of nerve cells and fibres.

This leads to irregular stimulation of the motor tracts. We do not as a rule get actual paralysis nor indeed the increased reflexes following secondary degeneration in the

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cord. We almost always find, however, some paresis, and one of the symptoms present in multiple sclerosis, mental dullness. Arndt affirms that there is always some mental disturbance in chorea. In chronic cases it is especially marked, as also in hereditary chorea.

In Mercier's paper on "Inhibition," in "Brain," October, 1888, he compares the nervous system to an army under its different officers; the loss of any one of these heads leads to disorder in some of the various departments. This idea has always seemed to me too mechanical. In the nervous system we have to do with living organisms; their action or function is continuous, and dependent on their nutrition. The higher centres are subject to constant influence from without, through their lower centres and nerve-paths, and this manner a constant relation or equilibrium of all the various parts of the nervous system is maintained. There is continuous action going on in the central ganglia, it is not only expended in the carrying out of some motor effect.

Let the peripheral stimuli become excessive, and these centres become exhausted, and as a result irregular efferent impulses are sent to the motor apparatus, as seen in the different occupation neuroses. I agree with him that no general or special inhibitory centre exists, but rather that the control or inhibition lies for each part in its corresponding centre.

An irritable condition of a centre, produced mechanically or through nutritional changes, may result in the so-called explosion of that centre, as in an epileptic seizure, or again, as in chorea, we may have continuous efferent impulses sent to the motor apparatus when no voluntary act is being carried out. When the lesion is more extreme, inhibition is still more affected, and purposive action only the more forcibly brings out the irregular motor effects.

It is possible, therefore, that lesions involving the nerve-tracts as well as the nerve-centres may be considered as causing chorea, thus allying it to multiple sclerosis; although when we remember that it is the muscles of specialized action—*i. e.*, of the hand—which are first and most

severely affected, it is probable that the primary lesion is in the cerebral centres controlling those parts.

In paralysis agitans we have a tremor which may be inhibited, and, if we accept Dr. Broadbent's theory of the seat of the lesion in this disease as lying in the muscular nerve-endings, we can readily understand the reason why this is so: the primary lesion points away from the cortex, whatever the secondary changes may be.

In acute chorea we find anæmia and vaso-motor changes more often present than any other condition.

Hanford, in "Brain," ———, 1889, quotes Dr. Dickinson as ascribing acute chorea to a widely spread hyperæmia of the nervous system, and in his own two cases he found, post-mortem, numerous hæmorrhages of the brain and cord with thrombosis and dilatation of the small vessels and capillaries. He considers the spinal cord, rather than the cerebral cortex, as the primary seat of the disease, as he believes the movements can be controlled in at least mild cases.

The connection of chorea and rheumatism is certainly more than accidental, but at the same time I do not believe it is as frequent as many writers would have us believe.

In chronic chorea we can look for atrophy of the cortex and degenerative changes in the nerve-tracts as a result of the hyperæmia and extravasations found in acute chorea. In support again of this theory I would refer to two cases reported by M. D. McLeod, in the "Journal of Mental Sciences," July, 1881, of two sisters, the chorea commencing, in the first, at the age of sixty; the autopsy revealed a cyst under the dura mater, over the left hemisphere, with flattening of the convolutions. The second sister was affected at the age of seventy, and the autopsy showed multiple tumors, with compression of the cortex. In these cases the compression and atrophy produced the chorea.

Dr. A. B. Ball, in an interesting paper on "Thrombosis of the Cerebral Sinuses and Veins," in the "Transactions of the Association of American Physicians," vol. iv., refers to this condition as occurring in chlorosis in young women, and reports several fatal cases with autopsies. The author

makes no reference to the occurrence of chorea in these cases, but that such a thrombosis may result in atrophy of the convolutions from permanent occlusion of the veins, entering the longitudinal sinus in the same manner as described by Gower, in cerebral spastic hemiplegia in children, seems to me very probable, although I cannot refer to any autopsies in proof of it.

Certain it is that in just such cases of chlorosis we find chorea unassociated with rheumatism or cardiac disease. The acute cases recover with the re-establishment of the circulation, while chronic chorea results from the atrophies referred to.

Acute chorea is as curable, therefore, as the conditions causing it, while in chronic chorea the prognosis depends on the character and extent of the lesion. The folly, therefore, of ascribing to the removal of a peripheral irritation, such as eye-strain, the possibility of effecting a cure in chronic chorea—that is, if we accept the pathology of it as just given—is apparent.

My paper has referred especially to the pathological changes in chronic chorea, and has emphasized the primary seat of the lesion as lying in the cortex cerebri.

UNILATERAL ABSENCE OF KNEE-JERK—PATHOLOGICAL FINDING IN A CASE.

Dr. A. Pick (Arch. f. Psych., 1889, vol. xx) reports a case in which a diagnosis of tabes and dementia paralytica was made. The symptoms were: girdle sensation, retention of urine and faeces, slight paresis of the left facial nerve about the mouth, unsteady gait, tremor of the tongue, delusions of grandeur, etc. The knee-jerk was absent on the left side, and only to be elicited by reinforcement upon the right.

Autopsy: Chronic internal haemorrhagic pacyhmeningitis, chronic inflammation with thickening of the other membranes, atrophy of the brain. Degeneration in the cord on the border of the columns of Goll and Burdach from the lower cervical region down. In the upper dorsal region there was on the left also a narrower stripe next to the posterior horn, and on the right a similar one in the middle of the column of Burdach. In the lower dorsal and in the lumbar portions of the cord, on the left side, the posterior root zone was affected. F. P.